

Relationship Between Aflatoxins and Hepatitis B Among Patients with Liver Disease at Moi Teaching and Referral Hospital, Uasin Gishu County

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Abstract

Liver disease is a major public health concern in Kenya, where hepatocellular carcinoma (HCC) is strongly associated with chronic hepatitis B virus (HBV) infection and dietary exposure to aflatoxins. The relationship between aflatoxin exposure and HBV remains insufficiently studied, limiting targeted prevention and management of liver disease. This study aims to establish relationship between aflatoxins and hepatitis B among patients with liver diseases at Moi Teaching and Referral Hospital, Uasin Gishu County. The study adopted a cross sectional descriptive study. Blood sample was collected in clot activated tubes for aflatoxin and hepatitis profile tests and the serum was separated and stored at -20°C for both male and female patients admitted in wards or visiting various clinics for diagnosis. Treatment or recovery regimens were selected through their case history and lab-diagnosis results of hepatitis profile, liver function tests (LFTs), AFP and histology. Conditional logistic regression was used to explore the relationship between aflatoxin B1-lysine and hepatitis B surface antigen titers in serum. Findings showed that the prevalence of Hepatitis B in Moi Teaching and Referral Hospital (17%) is higher than the national prevalence of 13.8%. The results suggest that older adults are more likely to be affected by Hepatitis B. However, the study found no significant difference in Hepatitis B prevalence between men and women. The results also showed mean value of 26.7289 pg/2g of aflatoxin exposure among the patients. Conditional logistic regression indicated that aflatoxin is associated with an increase in the log-odds of developing hepatitis B ($\chi^2 = 1.513$ and a p-value = 0.009, $p < 0.05$). This suggests that high levels of aflatoxin cause hepatitis B. Based on these findings, a pressing need exists to amplify public health campaigns that foster heightened awareness about both Hepatitis B and aflatoxin exposure. It is also imperative to establish standardized and routine screening protocols specifically aimed at identifying Hepatitis B among individuals presenting with symptoms of liver disease. Equally significant is the

proactive approach towards monitoring aflatoxin exposure among individuals displaying symptoms of liver disease. To this end, the implementation of systematic screening protocols tailored to aflatoxin exposure is paramount.

Keywords: Aflatoxin B1, Hepatitis B virus (HBV), Hepatocellular carcinoma (HCC), Liver disease, Aflatoxin-lysine adduct, Co-exposure, MTRH, Kenya

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Introduction

Liver disease is a major global health concern, ranging from acute hepatitis and chronic hepatitis to cirrhosis and hepatocellular carcinoma (HCC) (Younossi et al., 2023; Batool et al., 2022). Among the leading risk factors for progressive liver disease are chronic Hepatitis B Virus (HBV) infection and dietary exposure to aflatoxins, particularly aflatoxin B₁ (AFB₁) (Zhang et al., 2021; Melaram, 2021; Polpichai et al., 2024). Each of these factors independently contributes to liver damage, but evidence consistently shows that their interaction accelerates disease progression and worsens clinical outcomes (Chu et al., 2017; Moreno-León & Aguayo, 2025).

HBV is a DNA virus belonging to the Hepadnaviridae family that affects approximately 296 million people worldwide (WHO, 2021). It spreads primarily through contact with infected blood or body fluids, including perinatal transmission, unsafe medical injections, transfusions, and sexual contact (Inoue & Tanaka, 2016; Alsulaimany, 2023). Clinical outcomes range from asymptomatic

infection to acute hepatitis, and in chronic cases, persistent inflammation often leads to fibrosis, cirrhosis, hepatic decompensation, and HCC (Parikh et al., 2017; Songtanin et al., 2024). Integration of viral DNA into host genomes, along with the activity of viral proteins such as HBx, contributes to DNA repair disruption, genomic instability, and tumorigenesis (Mak & Kramvis, 2021). HBV is estimated to cause almost half of liver cancer cases globally, with the highest burden in sub-Saharan Africa and East Asia (Amponsah-Dacosta, 2021).

Aflatoxins, toxic metabolites primarily produced by *Aspergillus flavus* and *Aspergillus parasiticus*, contaminate staple foods such as maize, groundnuts, and rice in warm, humid regions (Fouad et al., 2019; Cary et al., 2018). The four most common types are B₁, B₂, G₁, and G₂, with AFB₁ being the most potent and classified as a Group 1 human carcinogen (IARC, 2012). AFB₁ is hepatotoxic, immunosuppressive, and mutagenic. After metabolic activation in the liver by cytochrome P450 enzymes, it forms AFB₁-

8,9-epoxide, a highly reactive compound that binds DNA and causes mutations, including the hallmark p53 codon 249 mutation (Kensler et al., 2011; Wild & Gong, 2010; Ferreira et al., 2019). Chronic exposure leads to fatty liver, necrosis, impaired regeneration, and progressive liver dysfunction, even before cancer develops (Fouad et al., 2019; Cary et al., 2018).

The relationship between aflatoxin exposure and HBV infection has been widely investigated in both experimental and human studies. Early animal experiments using woodchucks infected with the woodchuck hepatitis virus and HBV-transgenic mice provided critical evidence of a synergistic hepatocarcinogenic effect between AFB₁ and HBV infection (Suresh & Menne, 2021; Liu et al., 2019). These models demonstrated that HBV infection enhances the hepatotoxic effects of AFB₁ through upregulation of cytochrome P450 enzymes, leading to greater production of the mutagenic AFB₁-epoxide. HBV also impairs DNA repair and promotes oxidative stress, while aflatoxin metabolites directly damage DNA; together, these processes accelerate the progression from chronic hepatitis to cirrhosis and malignancy (Niu et al., 2021; Wangia, 2020; Capuozzo et al., 2021). At the molecular level, HBV infection alters hepatocyte function, making liver cells more vulnerable to aflatoxin-induced DNA damage (Kew, 2013). The HBV X protein (HBx) interferes with cell cycle regulation and DNA repair, further amplifying aflatoxin's carcinogenic impact (Groopman & Kensler, 2005). Epidemiological studies consistently confirm that individuals exposed to both HBV and aflatoxin have a much higher incidence of HCC compared to those exposed to either factor alone (Chen et al., 1996; Negash, 2018; Sagnelli et al., 2020). Biomarker studies reinforce this evidence,

with aflatoxin-albumin adducts and urinary aflatoxin metabolites strongly linked to advanced liver disease in HBV-positive individuals (Kumi, 2015). Globally, aflatoxin exposure is estimated to account for 4.6–28.2% of HCC cases, with the highest burden in regions where food contamination overlaps with high HBV prevalence (IARC, 1993; Mamo et al., 2020; Shephard, 2019). In Guatemala, AFB₁ levels were found to exceed the effect of HBV infection in some cases, indicating the strength of aflatoxin as a hepatotoxin (Alvarez et al., 2020), while in Ghana, HBV-positive patients with high aflatoxin biomarker levels show substantially higher odds of cirrhosis and HCC (Afum et al., 2016).

Despite this well-established global evidence, the interaction between chronic aflatoxin exposure and HBV infection has not been adequately studied in Kenya, where both risk factors are highly prevalent. Aflatoxin contamination is a recurring public health concern in the country; maize, the staple food, is the main exposure pathway (Awuor, 2022; Cherotich et al., 2025). Major outbreaks, including the severe 2004–2005 episode that resulted in 317 cases and 125 deaths after consumption of highly contaminated homegrown maize exceeding the national limit of 20 ppb, were associated with a toxigenic *Aspergillus flavus* S strain (Azziz-Baumgartner et al., 2005; Probst et al., 2007). Chronic exposure has been linked to HCC, particularly among individuals with high HBV prevalence (Mamo et al., 2020), and climatic conditions that favor fungal growth, poor farming and storage practices, and contamination of other foods such as milk continue to exacerbate the problem, disproportionately affecting children and low-income populations (Cherotich et al., 2025). While industrialized nations have largely minimized exposure through strict regulations (Guo et al., 2005), developing

countries continue to experience high contamination levels due to poor agricultural practices, weak food safety enforcement, and inadequate storage facilities, leaving more than 5 billion people at risk of chronic aflatoxin exposure (Shephard, 2003; Williams, Phillips et al., 2004).

Concurrently, HBV remains endemic in Kenya, with national prevalence estimated at 5–8%, classified as intermediate to high endemicity (Ochwoto et al., 2016). Transmission occurs through mother-to-child routes, unsafe medical procedures, sexual contact, and blood exposure. Western Kenya has a prevalence of 6–8%, while co-infection rates of 7.4% have been reported among HIV-positive populations in Nairobi and Kisumu (Akoth, 2021). The interaction between chronic aflatoxin exposure and HBV infection greatly increases the risk of HCC, yet this relationship has not been adequately characterized in the Kenyan context.

The issue is particularly important in Uasin Gishu County, one of the highest maize-producing regions in the country. Studies indicate that 19.4% of maize in the county is contaminated with AFB₁ (Osano et al., 2019), and the highest detections of mycotoxins nationally have been reported in this county (Okoth et al., 2012). Most patients with liver disease at Moi Teaching and Referral Hospital (MTRH), the main referral facility in the region, originate from this maize-growing area and are therefore highly vulnerable to aflatoxin exposure alongside HBV infection risks. The lack of adequate data on the combined effects of HBV infection and aflatoxin exposure among patients with liver disease limits effective prevention, diagnosis, and treatment strategies. This study therefore aimed to establish the

relationship between aflatoxins and hepatitis B among patients with liver diseases at MTRH, Uasin Gishu County. The specific objectives were to establish the prevalence of hepatitis B among liver disease patients attending MTRH, determine the prevalence of AFB₁ among these patients, and determine the relationship between AFB₁ and hepatitis B among liver disease patients attending MTRH.

Materials and Methods

Study Location and Design

This was a hospital-based cross-sectional analytical study conducted at Moi Teaching and Referral Hospital (MTRH) in Eldoret, Uasin Gishu County, Kenya. The study was carried out between January 2023 and June 2023. MTRH (Figure 1) is located at 0° 30' 44" North, 35° 16' 50" East and serves as Kenya's second-largest public referral hospital, with a catchment population of approximately 14 million people, representing about 40% of Kenya's total population. Uasin Gishu County is one of the leading maize-producing regions in Kenya, with the highest acreage under maize cultivation nationally, followed by Kakamega and Trans Nzoia counties (KNBS, 2021). The cross-sectional design was selected because it allows simultaneous assessment of exposure (aflatoxin) and outcome (HBV infection and liver disease status) within a defined population at a single point in time, enabling estimation of prevalence and identification of associations (Mekuria et al. (2023). However, this design limits inference regarding temporal sequence and causality.

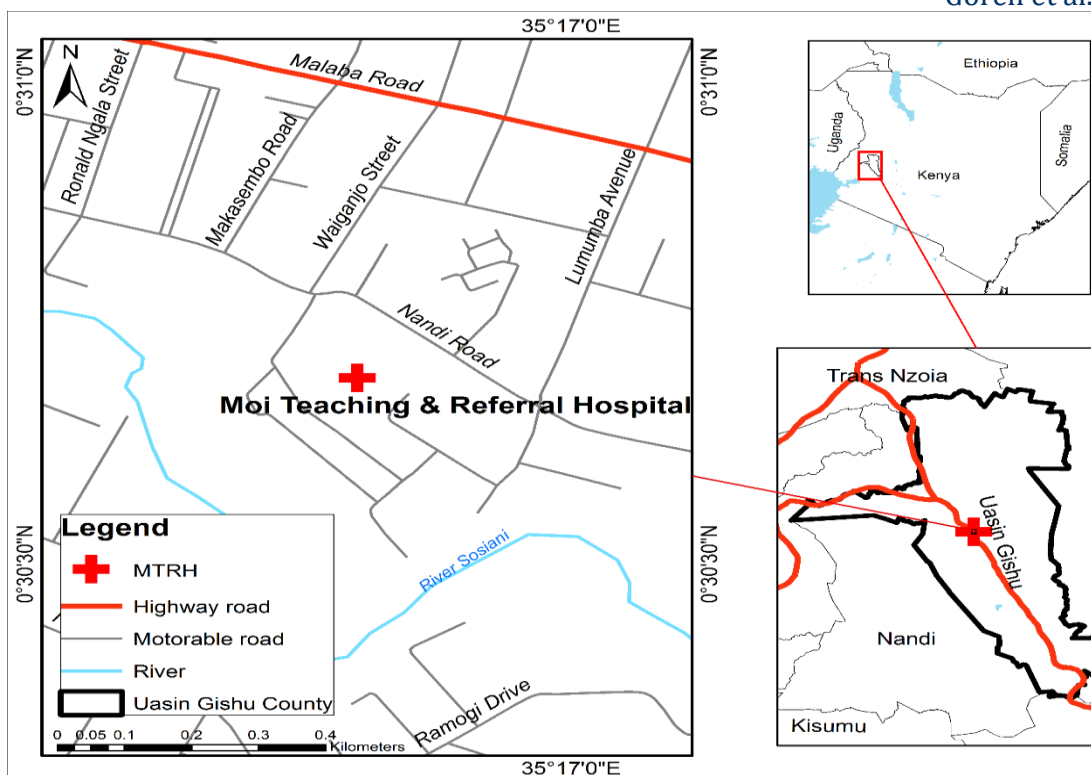


Figure 1: Map Showing the Location of the Study Area

Study Population, Sampling, and Selection Criteria

The target population comprised adult patients aged 18 to 70 years receiving care at MTRH for suspected chronic liver disease during the study period. Suspected chronic liver disease was defined as presentation with clinical features suggestive of chronic hepatic dysfunction (jaundice, hepatomegaly, ascites, or splenomegaly) accompanied by abnormal liver function tests (elevated alanine aminotransferase [ALT] or aspartate aminotransferase [AST] above the upper limit of normal, or low albumin) persisting for more than six months, or radiological evidence of chronic liver disease on abdominal ultrasound. The sample size was determined using Yamane's formula for finite populations:

$$n = \frac{N}{1 + Ne^2}$$

where n is the sample size, N is the target population (370 patients with suspected chronic liver disease attending MTRH

during the study period), and e is the precision level (0.05) at 95% confidence. This yielded a minimum sample size of 192. To account for an anticipated 10% non-response rate and incomplete laboratory results, the sample size was adjusted to 212 participants.

A census sampling technique was employed. All eligible patients presenting to the medical wards, hepatology clinic, or gastroenterology outpatient department at MTRH during the study period who met the inclusion criteria were approached for enrolment until the required sample size was attained.

Inclusion criteria: (i) Patients aged 18 to 70 years; (ii) Patients with a clinical diagnosis of chronic liver disease based on the criteria described above; (iii) Patients willing to provide informed consent.

Exclusion criteria: (i) Patients with secondary liver cancer (metastatic disease confirmed by imaging or histology); (ii) Patients who had undergone abdominal surgery within the preceding three

months; (iii) Patients with a documented diagnosis of asthma requiring regular bronchodilator or corticosteroid therapy; (iv) Patients with a history of alcohol abuse; (v) Patients on immunosuppressive therapy.

Laboratory Methods

Aflatoxin Biomarker Detection and Quantification

Serum samples were separated from blood and stored at -20°C until analysis. Aflatoxin exposure was assessed through the detection of serum aflatoxin-albumin adducts and quantification of aflatoxin B1-lysine (AFB1-lys) adducts. These biomarkers provide reliable indicators of internal aflatoxin burden, reflecting both recent and cumulative exposure.

For initial detection, enzyme-linked immunosorbent assays (ELISAs) were performed. Polystyrene microtiter plates were coated with aflatoxin B1-bovine serum albumin, followed by the addition of serum eluates and aflatoxin standards (25, 2.5, 0.25, 0.025, and 0.0 ng/ml). A monoclonal antibody solution was added to compete for binding, and after overnight incubation at 4°C , plates were washed with 0.05% Tween 20 in PBS. A secondary antibody was then applied for 2 h at room temperature, followed by additional washing with PBS and diethylamine buffer (pH 9). Substrate solution (4-nitrophenyl phosphate) was added, and absorbance was measured at 405 nm, allowing determination of aflatoxin-albumin adduct levels.

To strengthen reliability and quantify exposure with higher precision, competitive ELISAs for AFB1-lys adducts were carried out using AFB1-lys standards and monoclonal anti-AFB1-BSA antibodies. Plates were coated with AFB1-ovalbumin conjugate in carbonate buffer (pH 9.6) and incubated in the dark for 4 h.

Incubations with antibodies were performed for 2 h, with intermediate washes using PBS containing 0.1% Tween. Negative controls were included to check for non-specific binding. To validate ELISA results, samples were analyzed by HPLC with fluorescence detection. Samples were prepared by solid-phase extraction using C18 cartridges, followed by derivatization with trifluoroacetic acid. Chromatographic separation was performed on a reversed-phase C18 column (250×4.6 mm, $5\text{ }\mu\text{m}$ particle size) with a mobile phase of water: acetonitrile: methanol (60:25:15, v/v/v) at a flow rate of 1.0 mL/min. Fluorescence detection was set at excitation 399 nm and emission 457 nm. The HPLC method showed linearity ($R^2 = 0.998$) over the range 0.05–25 ng/mL, with recovery rates of 92–105% and intra-assay and inter-assay coefficients of variation (CV) of 4.2% and 6.8%, respectively.

Detection of hepatitis B surface antigen using ELISA

The procedure was done according to the manufacturer's kit (Hepatitis B Surface Antigen ELISA Kit" manufactured by various companies, such as Abcam, Bio-Rad, or Diazyme). Briefly, the micro-titre wells were fitted on the micro-titre plate and 50 μl of the sample, negative and positive controls and also the conjugate added to each well followed by incubation for one hour at 37°C . After incubation the wells were washed with wash buffer for six cycles. 50 μl of color reagent was added into each well and mixed gently then incubated for 15 min at 30°C and 100 μl stop solution added to each well. The absorbance was read using a spectrometer at a wavelength of 450 nm.

Quality control

Controls included two positive controls and two negative controls analyzed alongside batches of samples. A

plate was accepted if positive control CV was <15% and negative control absorbance was below 0.15. Any plate failing these criteria was repeated. Intra-assay CV for aflatoxin ELISA was 5.3%, and inter-assay CV was 8.1%. For HBsAg ELISA, intra-assay CV was 3.8% and inter-assay CV was 5.4%

Data analysis

Data was collected on a data entry form and later transferred to a computer database. Each entry form was labeled with a unique identifier, which is the participant number. After that, SPSS version 20 was used to evaluate the data. Odd ratios (ORs) between aflatoxin exposure and Hepatitis B virus were computed using conditional logistic regression. The association between serum hepatitis B surface antigen titers and aflatoxin B1-lysine adduct concentration was also investigated using conditional logistic regression.

Ethical consideration

Approval from the Institutional Research and Ethics Committee (IREC) and permission from Moi Teaching and Referral Hospital (MTRH) were obtained prior to commencing the study. Written informed consent was obtained from all participants before enrolment. For

participants who were illiterate, consent was obtained in the presence of an independent witness, with the information sheet read aloud in the participant's preferred language (English, Kiswahili, or Kalenjin). Participants were informed that participation was voluntary, and that withdrawal would not affect their clinical care.

Confidentiality was maintained by assigning each participant a unique alphanumeric code; names and personal identifiers did not appear on laboratory specimens or data collection forms. Data were stored in password-protected electronic files accessible only to the principal investigator and research assistant. Paper records were kept in locked cabinets. All data were used solely for academic purposes. Participants with abnormal laboratory findings were referred to the attending clinician for appropriate follow-up and management.

Results

Demographic Characteristics of Patients

The study examined gender and age distribution of patients who participated in the study. The findings are presented in Table 1.

Table 1: Patients’ Gender and Age Distribution

Variable	Category	Frequency	Percent
Gender	Female	97	50.5
	Male	95	49.5
	Total	192	100.0
Age Range	18–29 years	42	21.9
	30–39 years	47	24.5
	40–49 years	39	20.3
	50–59 years	36	18.8
	60 years & above	28	14.6
	Total	192	100.0

The gender distribution in Table 1 shows that out of 192 patients, 97 (50.5%)

were female and 95 (49.5%) were male. The sample was almost evenly divided,

with a slight female majority. Age distribution revealed that the largest proportion of patients (24.5%) were in the 30–39 years age group, followed closely by those aged 18–29 years (21.9%). About one-fifth (20.3%) were between 40–49 years, while 18.8% fell within the 50–59 years bracket. Patients aged 60 years and above accounted for 14.6% of the sample. This indicates that the majority of liver disease patients in the study were adults in their early to late thirties, with representation across all age groups.

The Prevalence of Hepatitis B Among Liver Disease Patients Attending MTRH

The study examined the prevalence of Hepatitis B virus (HBV) infection among patients presenting with liver disease symptoms at Moi Teaching and Referral Hospital (MTRH). The findings are presented in Figure 2.

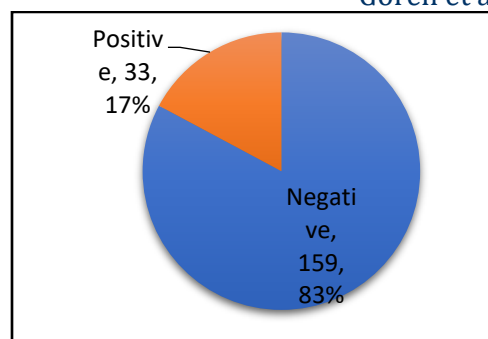


Figure 2: Prevalence of Hepatitis B among Liver Disease Patients Attending MTRH

The overall prevalence of hepatitis B among liver disease patients at MTRH was 17% (33 out of 192 patients), which exceeds the national prevalence estimate of 13.8%.

Prevalence of Hepatitis B in MTRH in Relation to Age and Gender

The study also evaluated the distribution of Hepatitis B virus (HBV) infection among liver disease patients at Moi Teaching and Referral Hospital (MTRH) based on age and gender. The findings are summarized in Table 2.

Table 2: Prevalence of Hepatitis B in MTRH in Relation to Age and Gender

Characteristic	Category	Negative n (%)	Positive n (%)	Total n (%)
Age (Years)	18–29	43 (93.5)	3 (6.5)	46 (100)
	30–39	38 (90.5)	4 (9.5)	42 (100)
	40–49	28 (71.8)	11 (28.2)	39 (100)
	50–59	26 (74.3)	9 (25.7)	35 (100)
	60 and above	24 (82.8)	5 (17.2)	29 (100)
Gender	Male	79 (83.2)	16 (16.8)	95 (100)
	Female	80 (82.5)	17 (17.5)	97 (100)
Total		159 (82.8)	33 (17.2)	192 (100)

The overall prevalence of Hepatitis B among patients at Moi Teaching and Referral Hospital was 17%. The highest rates were recorded among individuals in their 40s and 50s, while the lowest occurred in the 18–29 age group. A decrease was also noted among patients aged 60 years and above. Prevalence between male and female patients was almost identical.

The Prevalence of Aflatoxin among Patients Attending MTRH Presenting Liver Disease Symptoms

Table 3 shows the prevalence of aflatoxin (AF-alb) among patients attending Moi Teaching and Referral Hospital (MTRH) presenting with liver disease symptoms. The statistics include the number of patients (N), minimum and maximum values, mean, standard

deviation (Std. Deviation), skewness, and kurtosis.

Table 3: Prevalence of Aflatoxin Among Patients Attending MTRH

Parameter	AF-alb (pg/2g o1 albu2in)
N	192
Minimum	9.91
Maximum	50.82
Mean	26.7289
Std. Deviation	7.72952
Skewness	0.389
Kurtosis	0.205

The AF-alb levels ranged from a minimum of 9.91 pg/2g of albumin to a

maximum of 50.82 pg/2g, with a mean of 26.73 pg/2g and a standard deviation of 7.73, showing wide variability in exposure levels. The distribution of aflatoxin levels showed a slight positive skewness (0.389), indicating a mild tilt towards higher exposure values, while the kurtosis value of 0.205 suggested that the distribution was approximately normal.

Prevalence of Aflatoxin Exposure in Relation to Age and Gender

The findings were further analyzed in relation to age and gender to establish distribution patterns across subgroups as indicated in Table 4.

Table 4: Prevalence of Aflatoxin among Patients Attending MTRH in Relation to Age and Gender

Characteristic	Test	N	Mean	Standard Deviation	F	p value
Age	Negative	159	42.83	15.60	4.78	0.03
	Positive	33	49.50	13.98		
	Total	192	43.87	15.51		
Gender	Negative	159	1.49	0.50	0.00	0.95
	Positive	33	1.50	0.51		
	Total	192	1.49	0.50		

The study findings indicate a significant disparity in Hepatitis B prevalence across different age groups. Specifically, the mean age of individuals who tested positive for Hepatitis B was 49.5 years, while the mean age of those who tested negative was 42.8 years. This difference is statistically significant, with a p-value of 0.03. However, no significant difference was observed between men and women, as the prevalence rates were nearly identical, with a p-value of 0.95 showing that gender was not associated with Hepatitis B infection in this study population.

Association between Liver Function Tests and Aflatoxin BI

The results in Table 5 presents the correlation coefficients between various liver function tests (ALB, ALP, GGT, AST, ALT, TP) and their association with aflatoxin exposure. The correlation coefficients measure the strength and direction of the linear relationship between two variables. The significance levels are also provided to determine if the correlations are statistically significant.

Several significant relationships between liver symptoms and biochemical indicators were found by the correlation analysis. First, serum albumin (ALB) and total protein (TP) showed a strong positive connection ($r = 0.593^{**}$, $p < 0.01$).

Table 5: Association between liver function tests and aflatoxin

Test	Statistic	ALB	ALP	GGT	AST	ALT	TP
ALB	Pearson						
	Correlation	1					
	Sig. (2-tailed)						
ALP	Pearson						
	Correlation	-.230*	1				
	Sig. (2-tailed)	0.037					
GGT	Pearson						
	Correlation	-0.021	.381**	1			
	Sig. (2-tailed)	0.851	0.000				
AST	Pearson						
	Correlation	0.051	-0.001	-0.029	1		
	Sig. (2-tailed)	0.644	0.989	0.791			
ALT	Pearson						
	Correlation	0.159*	-0.013	-0.037	.844**	1	
	Sig. (2-tailed)	0.045	0.907	0.739	0		
TP	Pearson						
	Correlation	.593**	-0.133	0.066	0.003	0.06	1
	Sig. (2-tailed)	0.000	0.23	0.551	0.978	0.584	

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

Key:

ALP-Alkaline Phosphatase

ALT-Alanine Transaminase

ALB-Albumin Test

AST-Aspartate Aminotransferase Test

GGT-Gamma-Glutamyltransferase

ALB-Albumin

TP-Total Protein

This implies that there is a correlation between greater TP levels and higher ALB levels. Alkaline phosphatase (ALP) and ALB, on the other hand, showed a strong negative connection ($r = -0.230^*$, $p < 0.05$), suggesting that lower ALB levels are linked to greater ALP levels. Furthermore, there was a noteworthy positive connection between TP and alanine aminotransferase (ALT) ($r = 0.844^{**}$, $p < 0.01$), suggesting that greater TP levels are linked to higher ALT levels. No significant correlations were

identified between gamma-glutamyl transferase (GGT) or aspartate aminotransferase (AST) and any other liver symptoms. TP exhibited weak positive correlations with GGT and weak negative correlations with ALP, though these were not statistically significant.

Relationship between Aflatoxin BI and Hepatitis B

Table 6 shows the results of a regression analysis examining relationship between

aflatoxin and hepatitis B. The analysis revealed a significant relationship between aflatoxin and hepatitis ($\chi^2 = 1.513$ and a p-value = 0.009, $p < 0.05$). This suggests that aflatoxin impact the development of hepatitis B. When considering the change from the previous step, the chi-square value was $\chi^2 = 1.557$ with a p-value of 0.212. This indicates that

adding additional factors did not significantly alter the relationship between aflatoxin and hepatitis. However, the change from the previous block showed a chi-square value of $\chi^2 = 1.557$ and a p-value of 0.012 ($p < 0.05$), suggesting that the interaction between aflatoxin and hepatitis remains relevant in different contexts.

Table 6: Omnibus Tests of Model Coefficients

-2 Log Likelihood	Overall (score)			Change From Previous Step	Change From Previous Block				
	Chi-square	Df	Sig.	Chi-square	Df	Sig.	Chi-square	df	Sig.
287.167	1.513	1	0.009	1.557	1	0.212	1.557	1	0.012

Table 7 outlines the variables included in the equation as well as their corresponding coefficients and statistical measures. Specifically, it focuses on the variable "ALB" (Albumin levels) and its potential significance in the context of the analysis. The coefficient B for the variable ALB is 0.029, with a standard error (SE) of 0.024. This indicates the change in the log-odds of the outcome (hepatitis B) for a one-unit change in Albumin levels. In this case, a positive coefficient suggests that an increase in Albumin levels is associated with an increase in the log-odds of hepatitis B.

the outcome (hepatitis B.). The Exp(B) value is 1.971, indicating the odds ratio associated with a one-unit increase in Albumin levels. Since the value is more than 1, it suggests that higher Albumin levels are associated with a higher odds ratio of developing hepatitis B. The results suggest that Albumin levels (ALB) are statistically significant in the equation when considering their potential impact on the development of hepatitis B. The positive coefficient and odds ratio imply that higher Albumin levels may be associated with increased likelihood of developing hepatitis B.

Table 7: Variables in the Equation

Variables in the Equation						
	B	SE	Wald	d	Sig.	Exp(B)
			d	f		
AL	0.0	0.0	1.5	1	0.0	1.97
B	29	24	12		19	1

The Wald statistic measures the significance of the coefficient, and for the variable ALB, it has a value of 1.512 with 1 degree of freedom, resulting in a significance level (Sig.) of 0.019. This level of significance indicates that the variable ALB is statistically significant in relation to

Discussion

The results (Figure 1) revealed that the prevalence of Hepatitis B in Moi Teaching and Referral Hospital (17%) is higher than the national prevalence of 13.8%. These results align with the findings of Makokha et al. (2023), who reported an overall pooled prevalence estimate of HBV in Kenya at 7.8% yet it surpasses the prevalence in certain regions of Kenya, such as Western Kenya (8.6%). Additionally, Wahome et al. (2022) documented a sero-positivity rate of 8.8% for HBsAg, indicative of a significant proportion of individuals harboring HBV in

the population. Jepkemei et al. (2020) conducted a study specifically in Western Kenya, uncovering a lower seroprevalence of 3.4% for HBsAg among school-going adolescent donors aged 18 to 25 years. These regional variations in prevalence rates confirm the disparities observed in Table 2, where Western Kenya demonstrates a comparatively lower prevalence than the national average and the prevalence at Moi Teaching and Referral Hospital. Downs et al. (2020) contributed to understanding HBV prevalence by categorizing studies into different risk groups and evaluating prevalence rates within each group. Their findings demonstrate significant variability in prevalence rates, ranging from 3.4% to 29.2% across different risk categories. Several factors may contribute to these regional differences in Hepatitis B prevalence. Environmental and socioeconomic factors, as well as variations in healthcare access and quality, can influence the spread and detection of Hepatitis B within different regions. Moreover, differences in study populations and methodologies may also contribute to the observed variations in prevalence rates (Zampino, et al., 2015).

The study findings revealed that the prevalence of aflatoxin exposure among the patients was 26.7289 pg/2g. (Table 3). This aligns with similar observations reported globally. For instance, Chen et al. (2022) estimated aflatoxin exposure levels between 17–30 ng/kg body weight/day in China. In Pakistan, Zaterka and Eisig (2016) highlighted a high prevalence of aflatoxins in food and feed commodities, with levels varying widely across different products. Similarly, Davwar et al. (2019) found median aflatoxin M1 levels of 177.3 pg/ml in North-Central Nigeria, with significant associations between aflatoxin exposure and liver fibrosis scores. In the Ashanti region of Ghana, Afum et al. (2016)

reported widespread aflatoxin exposure, underscoring the global prevalence of this issue. In Kenya, Mekuria et al. (2023) documented high detection rates of F-alb levels in Eastern Ethiopia, which were significantly associated with liver disease. Another Kenyan study by Githanga (2019) detected AFB1 in 100% of the children studied, indicating notable implications for anti-HBs levels. Furthermore, Kimani et al. (2019) reported significant associations between serum HBsAg levels and aflatoxin exposure in case subjects, emphasizing the complex interplay between aflatoxin exposure and liver disease markers. These findings underscore the pervasive nature of aflatoxin exposure across different regions and its significant association with liver disease symptoms. The data from MTRH contribute to the growing body of evidence that highlights the global public health challenge posed by aflatoxin contamination.

The study suggests that aflatoxin increases the risk of developing of Hepatitis B. With a p-value of 0.212, the addition of other factors did not significantly alter the relationship between aflatoxin and hepatitis B (Table 6). However, a subsequent p-value of 0.012 ($p < 0.05$) indicates that the interaction between aflatoxin and hepatitis remains relevant in different contexts. The findings from this study suggest a notable interaction between aflatoxin exposure and hepatitis B. These findings agree with several studies that support the synergistic effects of aflatoxin exposure and hepatitis viruses in liver carcinogenesis. For instance, Liu and Wu (2010) reported that aflatoxin B1 (AFB1), a potent carcinogen, exacerbates the effects of chronic hepatitis B virus (HBV) infection.

Similarly, Davwar et al. (2019) found median aflatoxin M1 levels in North-Central Nigeria with significant associations between aflatoxin exposure and liver fibrosis scores, which aligns with

our results. Mekuria et al. (2023) documented high detection rates of aflatoxin in Eastern Ethiopia, significantly associated with liver disease, further corroborating our findings on the prevalence and impact of aflatoxin exposure. Additionally, Kimani et al. (2019) reported significant associations between serum HBsAg levels and aflatoxin exposure, emphasizing the complex interplay between aflatoxin exposure and liver disease markers, which supports our conclusion. However, the findings disagree with some studies that present differing views on the interaction between aflatoxin and hepatitis. Chen et al. (2022) estimated aflatoxin exposure levels in China and suggested that while aflatoxin is a significant health concern, its interaction with hepatitis in developing HCC may not be as pronounced as previously thought, indicating other factors might also play a crucial role. Zaterka and Eisig (2016) highlighted the high prevalence of aflatoxins in Pakistan but did not find a strong association between aflatoxin exposure and liver disease progression when hepatitis was also considered, suggesting regional variations. Similarly, Afum et al. (2016) reported widespread aflatoxin exposure in Ghana but did not find a significant link between aflatoxin levels and hepatitis, indicating that socioeconomic factors and overall nutrition might modulate the effects of aflatoxin on liver disease progression.

Despite these differing views, there is a consensus on the significant health risks posed by aflatoxin. Wild and Turner (2002) argued for the incorporation of aflatoxin testing in clinical settings, suggesting that measuring aflatoxin levels in blood or urine can help assess exposure and mitigate further liver damage. This approach aligns with the recommendations from our study, highlighting the importance of aflatoxin testing in healthcare protocols to better

manage and prevent liver diseases. Overall, the interaction between aflatoxin exposure and hepatitis B, as evidenced by this study and supported by several prior research studies. These findings are consistent with studies conducted by Cao and Wang (2014) and Wang et al. (2023) in China, which demonstrated significant alterations in serum biochemical indices due to AFB1 exposure, highlighting the importance of understanding aflatoxin's impact on liver health across different populations. Additionally, studies by Githang'a (2019), Kimani et al. (2019), and other relevant literature have also highlighted the associations between aflatoxin and Hepatitis B.

Conclusion

The findings indicated that Hepatitis B infection is a significant contributor among patients presenting with liver disease symptoms at Moi Teaching and Referral Hospital (MTRH), implying that viral etiology plays a substantial role in the observed liver disease burden within this population. The findings highlight the presence of considerable aflatoxin exposure among the patients, suggesting its potential role in liver disease progression. Results from conditional regression analysis indicated that higher levels of aflatoxin exposure contribute to the development of Hepatitis B. This finding demonstrates that aflatoxin exposure significantly increases the risk of Hepatitis B infection.

Recommendations

1. Regular testing of maize at farms, storage facilities, and markets will help identify contamination early and protect consumers.
2. To address the relationship between aflatoxin exposure and Hepatitis B, advocating for stricter food safety regulations is paramount. This includes

enforcing maximum allowable limits for aflatoxin in food products, conducting regular monitoring activities and ensuring compliance with these standards.

3. Supporting research into innovative technologies and practices for reducing aflatoxin contamination is also vital. Investments in new solutions, tailored to local conditions, will provide effective strategies for managing aflatoxin risks.
4. Expand the age range of study participants to include individuals younger than 18 years and older than 70 years to assess the impact of AFB1 exposure across different age groups. This would provide insights into age-specific susceptibility to AFB1-related liver diseases.
5. Further research is needed to geographically map the origin of patients, which would facilitate the identification of high-risk areas for aflatoxin exposure.

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